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* * * * * STN Columbus * * * * *

FILE 'HOME' ENTERED AT 14:25:22 ON 29 JUN 2005

=> file medline

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.63	0.63

FILE 'MEDLINE' ENTERED AT 14:27:18 ON 29 JUN 2005

FILE LAST UPDATED: 28 JUN 2005 (20050628/UP). FILE COVERS 1950 TO DATE.

On December 19, 2004, the 2005 MeSH terms were loaded.

The MEDLINE reload for 2005 is now available. For details enter HELP RLOAD at an arrow prompt (=>). See also:

<http://www.nlm.nih.gov/mesh/>
http://www.nlm.nih.gov/pubs/techbull/nd04/nd04_mesh.html

OLDMEDLINE now back to 1950.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2005 vocabulary.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s (inhibit delay) (P) (cytokinesis cytodieresis interphase) and (chemotherapeutic anticancer antineoplastic) and (cytochalasin dihydroxytochalasin jasplakinolide chondramide latrunculin)

138193 INHIBIT
63618 DELAY
12 INHIBIT DELAY
(INHIBIT (W) DELAY)
3488 CYTOKINESIS
10 CYTODIERESIS
12634 INTERPHASE
0 CYTOKINESIS CYTODIERESIS INTERPHASE
(CYTOKINESIS (W) CYTODIERESIS (W) INTERPHASE)
0 (INHIBIT DELAY) (P) (CYTOKINESIS CYTODIERESIS INTERPHASE)
19947 CHEMOTHERAPEUTIC
17245 ANTICANCER
187866 ANTINEOPLASTIC
0 CHEMOTHERAPEUTIC ANTICANCER ANTINEOPLASTIC
(CHEMOTHERAPEUTIC (W) ANTICANCER (W) ANTINEOPLASTIC)
9841 CYTOCHALASIN
0 DIHYDROXYTOCHALASIN
240 JASPLAKINOLIDE
6 CHONDRAMIDE
602 LATRUNCULIN
0 CYTOCHALASIN DIHYDROXYTOCHALASIN JASPLAKINOLIDE CHONDRAMIDE
LATRUNCULIN
(CYTOCHALASIN (W) DIHYDROXYTOCHALASIN (W) JASPLAKINOLIDE (W) CHONDRAMIDE (W) LATRUNCULIN)
L1 0 (INHIBIT DELAY) (P) (CYTOKINESIS CYTODIERESIS INTERPHASE) AND
(CHEMOTHERAPEUTIC ANTICANCER ANTINEOPLASTIC) AND (CYTOCHALASIN
DIHYDROXYTOCHALASIN JASPLAKINOLIDE CHONDRAMIDE LATRUNCULIN)

=> s (inhibit delay) (P) (cytokinesis cytodieresis interphase) and (cytochalasin dihydroxytochalasin jasplakinolide chondramide latrunculin)

```

138193 INHIBIT
63618 DELAY
  12 INHIBIT DELAY
      (INHIBIT(W)DELAY)
3488 CYTOKINESIS
  10 CYTODIERESIS
12634 INTERPHASE
  0 CYTOKINESIS CYTODIERESIS INTERPHASE
      (CYTOKINESIS(W)CYTODIERESIS(W)INTERPHASE)
  0 (INHIBIT DELAY) (P) (CYTOKINESIS CYTODIERESIS INTERPHASE)
9841 CYTOCHALASIN
  0 DIHYDROXYTOCHALASIN
  240 JASPLAKINOLIDE
  6 CHONDRAMIDE
  602 LATRUNCULIN
  0 CYTOCHALASIN DIHYDROXYTOCHALASIN JASPLAKINOLIDE CHONDRAMIDE
      LATRUNCULIN
      (CYTOCHALASIN(W)DIHYDROXYTOCHALASIN(W)JASPLAKINOLIDE(W)CHONDR
      AMIDE(W)LATRUNCULIN)
L2      0 (INHIBIT DELAY) (P) (CYTOKINESIS CYTODIERESIS INTERPHASE) AND
      (CYTOCHALASIN DIHYDROXYTOCHALASIN JASPLAKINOLIDE CHONDRAMIDE
      LATRUNCULIN)

```

=> s (inhibit delay) (P) (cytokinesis cytodieresis interphase) and "cytochalasin dihydroxytochalasin jasplakinolide chondramide latrunculin)
 MISMATCHED QUOTE 'AND "CYTOCHALAS'
 Quotation marks (or apostrophes) must be used in pairs,
 one before and one after the expression you are setting
 off or masking.

```

=> s inhibit or delay
      138193 INHIBIT
      63618 DELAY
L3      200566 INHIBIT OR DELAY

```

```

=> l3 (P) cytokinesis
L3 IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

```

```

=> l3(P)cytokinesis
L3(P)CYTOKINESIS IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

```

```

=> l3 and cytokesesis
L3 IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

```

```

=> s l3 (P) cytokinesis
      3488 CYTOKINESIS
L4      206 L3 (P) CYTOKINESIS

```

```

=> s l4 and cytochalasin
      9841 CYTOCHALASIN
L5      23 L4 AND CYTOCHALASIN

```

=> d 20-23 bib abs

```

L5      ANSWER 20 OF 23      MEDLINE on STN
AN      81168410      MEDLINE

```

DN PubMed ID: 6111566
 TI A permeabilized cell model for studying cell division: a comparison of anaphase chromosome movement and cleavage furrow constriction in lysed PtK1 cells.
 AU Cande W Z; McDonald K; Meeusen R L
 NC BRSI 507 RR0706 (NCRR)
 GM 23238 (NIGMS)
 SO Journal of cell biology, (1981 Mar) 88 (3) 618-29.
 Journal code: 0375356. ISSN: 0021-9525.
 CY United States
 DT Journal; Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals
 EM 198106
 ED Entered STN: 19900316
 Last Updated on STN: 19970203
 Entered Medline: 19810613
 AB After lysis in a Brij 58-polyethylene glycol medium, PtK1 cells are permeable to small molecules, such as erythrosin B, and to proteins, such as rhodamine-labeled FAB, myosin subfragment-1, and tubulin. Holes are present in the plasma membrane, and the mitochondria are swollen and distorted, but other membrane-bounded organelles of the lysed cell model are not noticeably altered. After lysis, the mitotic apparatus is functional; chromosomes move poleward and the spindle elongates. Cells lysed while in **cytokinesis** will continue to divide for several minutes. Addition of crude tubulin extracts, MAP-free tubulin, or taxol to the lysis medium retards anaphase chromosome movements but does not affect cleavage. On the other hand, N-ethylmaleimide-modified myosin subfragment-1, phalloidin, and **cytochalasin B** inhibit cleavage but have no effect on anaphase chromosome movements under identical lysis conditions. These results suggest that actomyosin plays no functional role in anaphase chromosome movement in mammalian tissue culture cells and that microtubule depolymerization is a rate-limiting step for chromosome-to-pole movements.

L5 ANSWER 21 OF 23 MEDLINE on STN
 AN 80134838 MEDLINE
 DN PubMed ID: 6444598
 TI Ultrastructural changes in the embryonic cells of the frog *Microhyla ornata* after **cytochalasin H** treatment.
 AU Wadekar G; Dastur D K; Mulherkar L
 SO Experimental cell biology, (1980) 48 (2) 155-66.
 Journal code: 7701827. ISSN: 0304-3568.
 CY Switzerland
 DT Journal; Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals
 EM 198005
 ED Entered STN: 19900315
 Last Updated on STN: 19900315
 Entered Medline: 19800523
 AB Biological effects of **cytochalasin H** (CH), a newly isolated mould metabolite, have been found to bring about disaggregation of embryonic cells and to **inhibit cytokinesis**. Disaggregation is known to be a phenomenon related to the cell surface. (The cells are held together by a mucopolysaccharide glycoprotein complex.) In the present work the fact that the mucopolysaccharide glycoprotein surface coat gets affected by CH treatment is confirmed by electron microscopy with the help of Lanthanum, a specific marker, which gets selectively absorbed to the cell surface material and renders it electron dense. The ultrastructural observations indicated the reduction of the cell surface material in treated embryos as compared to the controls. The reappearance of lanthanum-bound cell surface material in the recovered embryos was also observed. However, the exact mechanism of the action of CH on the cell surface remains to be clarified.

L5 ANSWER 22 OF 23 MEDLINE on STN
 AN 77235913 MEDLINE
 DN PubMed ID: 882839
 TI **Cytochalasin B** partially inhibits the oxalate-induced radial segmentation of mononucleated blood cells.
 AU Simmingskold G; Rydgren L; Norberg B; Soderstrom U B; Ponten J
 SO Scandinavian journal of haematology, (1977 Jul) 19 (1) 33-8.
 Journal code: 0404507. ISSN: 0036-553X.
 CY Denmark
 DT Journal; Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals
 EM 197709
 ED Entered STN: 19900314
 Last Updated on STN: 19900314
 Entered Medline: 19770917
 AB **Cytochalasin B** (CB), 5 microgram/ml (= 1.0×10^{-5} M), inhibited the oxalate-induced radial segmentation of the nuclei of lymphocytes and monocytes from peripheral blood. The median inhibition was 60%. The oxalate-induced radial segmentation (RS) is thought to be due to a microtubule-dependent contraction of the intermitotic residue of the mitotic apparatus around the nucleus. CB is thought to **inhibit** cell locomotion and **cytokinesis** by a centripetal contraction of the membrane-associated contractile cell layer without subsequent relaxation. It is thus suggested that the CB inhibition of the oxalate-induced RS was due to a spatial interference of the CB-induced contraction with the formation of RS nuclei.

L5 ANSWER 23 OF 23 MEDLINE on STN
 AN 76027739 MEDLINE
 DN PubMed ID: 1165581
 TI Membrane effects of **cytochalasin B**. Competitive inhibition of facilitated diffusion processes in rat hepatoma cells and other cell lines and effect on formation of functional transport sites.
 AU Plagemann P G; Zylka J H; Erbe J; Estensen R D
 SO Journal of membrane biology, (1975 Aug 11) 23 (1) 77-90.
 Journal code: 0211301. ISSN: 0022-2631.
 CY United States
 DT Journal; Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals
 EM 197512
 ED Entered STN: 19900313
 Last Updated on STN: 19970203
 Entered Medline: 19751212
 AB **Cytochalasin B** competitively inhibits the transport of 2-deoxy-D-glucose and thymidine in a number of different cell lines (Novikoff rat hepatoma cells, mouse L, S180 and Ki-MSV-transformed BALB/3T3 cells, and human HeLa cells). The apparent Km values for the transport of these substrates as well as the apparent Ki values for the inhibition by **cytochalasin B** are very similar for the various cell lines, and the effect is readily and completely reversed by removal of the chemical. Thymidine transport by Chinese hamster ovary cells however, is little affected by **cytochalasin B**, whereas the transport of 2-deoxy-D-glucose, uridine and guanine by these cells is competitively inhibited to about the same extent as in other cell lines. In addition and concomitant with the inhibition of **cytokinesis** and an alteration in cell shape, **cytochalasin B** also impairs and delays the formation of functional transport sites for thymidine, guanine and choline in synchronized populations of Novikoff cells without affecting the apparent affinities of the transport systems for their substrates. This effect is unrelated to the direct inhibition of the transport processes, since the drug does not directly **inhibit** choline transport and has no effect on the formation of 2-deoxy-D-glucose

transport sites in spite of the fact that it strongly inhibits the transport of this substrate. The inhibition of functional transport sites may be due to the induction of a structural alteration in the membrane by **cytochalasin B** which impairs the insertion of new proteins of certain but not all transport systems into the membrane.

=> s l4 and cytochalasin and chemotherapeutic

9841 CYTOCHALASIN

19947 CHEMOTHERAPEUTIC

L6 1 L4 AND CYTOCHALASIN AND CHEMOTHERAPEUTIC

=> d 1 abs bib

L6 ANSWER 1 OF 1 MEDLINE on STN

AB INTRODUCTION: The in vitro chemosensitivity testing aims at predicting the response of an individual tumour to chemotherapy choosing optimal agents for a particular patient. Among many chemosensitivity tests developed over the years [1-6], special emphasis was made on clonogenic assays that showed good use and correlation between laboratory and clinical data [7-9]. One of the assays used to predict the response to various anti-cancer modalities is the micronucleus assay using the **cytokinesis-block** [12-14]. This block is achieved by administration of **Cytochalasin-B** in order to prevent cytoplasmic, but not the nuclear, division. This leads to micronucleus formation which are counted in binuclear cells. Since there are only a few reports of the use of this assay in predicting chemosensitivity [13, 16], we explored the possibility of using this assay to predict chemosensitivity to various anti-cancer agents. MATERIAL AND METHODS: Exponentially growing SCC VII cells were treated with various concentrations of 11 anti-cancer agents: Mitomycin C, Doxorubicin (ADR), Epirubicin (EPI), Cisplatin, Carboplatin (CBDCA), Etoposide (VP-16), Vincristine, 5-fluorouracil, Methotrexate, Nimustine, and Dacarbazine for 1 hour. After that, **Cytochalasin-B** was added and dishes were incubated. After various time intervals, cells were fixed in situ and dried. Electron microscope was used to count the number of micronuclei (MN) in binucleate cells as well as multinucleate cells (MNC) in the total cell population. Cell survival was also evaluated by using the colony formation assay [18]. RESULTS: Maximal % of binucleate cells (BNC) was usually reached at 24-30 hours of culture, except for cells treated with ADR and EPI, in which it was reached at 30-72 hours (Figures 1 and 2). All drugs induced formation of micronuclei and dose-response curves for micronucleus frequency were obtained using the data at peak % BNC times. For all drugs, micronucleus frequency increased with concentration (Figure 3), but at the highest concentration used (considered to be overly toxic-Figure 4), the micronucleus frequency was rather lower. This decrease in micronucleus frequency was largely attributed to the decrease in % BNC. When the data at the highest concentrations of all drugs were excluded, a correlation was found between micronucleus frequency and surviving fraction ($r = 0.85$; $p < 0.001$) (Figure 5). DISCUSSION: Since micronucleus formation is a sign of chromosome damage that leads to cell death, we used this assay to evaluate chemosensitivity in 11 widely used anticancer agents. Although they can be classified according to mechanism of action as different class agents, they have in common the formation of micronuclei as a sign of cytotoxicity. Cell cycle arrest observed in some agents might be evaluated by assessing the **delay** in increase of BNC and MNC. The difference observed regarding cell cycle arrest suggested different mechanisms of its action. MN frequency was almost dose-dependent at lower concentrations, but at the highest concentrations, it obviously decreased, showing, therefore, some discrepancies with the data obtained when radiosensitivity was tested that way [14], probably due to extreme toxicity of agents. The optimal concentrations seem to be those providing a 20-80% surviving fraction. Another slight difference, when compared with similar radiosensitivity studies is a decrease with longer duration of culture observed in chemosensitivity testings. The

reason for this difference is still unknown, but it emphasized the necessity for choosing the optimal duration of culture, probably that necessary for reaching maximal % BNC. This assay seems useful in predicting chemosensitivity of at least some tumour cells to various (appropriate) concentrations of various anti-cancer agents. However, new studies are warranted to further use of this assay, before testing it in clinical practice.

AN 97206774 MEDLINE
DN PubMed ID: 9102841
TI [Significance of formation of micronuclei in SCC VII murine cells treated with various **chemotherapeutic** agents].
Znacaj formiranja mikronukleusa u murinim celijama SCC VII tretiranih razlicitim hemioterapijskim agensima.
AU Jeremic B; Sibamoto J; Abe M
CS Department of Oncology, Clinical-Hospital Centre, Kragujevac, Yugoslavia.
SO Srpski arhiv za celokupno lekarstvo, (1996 Jul-Aug) 124 (7-8) 169-74.
Journal code: 0027440. ISSN: 0370-8179.
CY Yugoslavia
DT Journal; Article; (JOURNAL ARTICLE)
LA Serbian
FS Priority Journals
EM 199704
ED Entered STN: 19970424
Last Updated on STN: 19970424
Entered Medline: 19970416

=>

---Logging off of STN---

=>

Executing the logoff script...

=> LOG Y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	5.28	5.91

STN INTERNATIONAL LOGOFF AT 14:33:42 ON 29 JUN 2005

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FILE 'HOME' ENTERED AT 18:29:37 ON 29 JUN 2005

=> file medline

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

SESSION

FULL ESTIMATED COST

0.21

0.21

FILE 'MEDLINE' ENTERED AT 18:29:57 ON 29 JUN 2005

FILE LAST UPDATED: 29 JUN 2005 (20050629/UP). FILE COVERS 1950 TO DATE.

On December 19, 2004, the 2005 MeSH terms were loaded.

The MEDLINE reload for 2005 is now available. For details enter HELP
RLOAD at an arrow prompt (=>). See also:

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OLDMEDLINE now back to 1950.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the
MeSH 2005 vocabulary.

This file contains CAS Registry Numbers for easy and accurate
substance identification.

=> s trifluorothymidine cytarabine 6-thioguanine 6-mercaptoputrine gemcytabine
fludarabine floxuridine ftorafur methotrexate trimetrexate raltitrexed edatrexate
lometrexol hydroxyurea vincristine vinblastine vinorelbin vindesine paclitaxel
docetaxel irinotecan topotecan "9-amino-s(20)-camptothecine"
PREVIOUS MSG TOO LONG

---Logging off of STN---

=>
Executing the logoff script...

=> LOG Y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	1.14	1.35

STN INTERNATIONAL LOGOFF AT 18:32:01 ON 29 JUN 2005

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LOGINID:SSPTAMXG1614

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FILE 'HOME' ENTERED AT 18:36:28 ON 29 JUN 2005

=> index bioscience

FILE 'DRUGMONOG' ACCESS NOT AUTHORIZED

COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
0.21	0.21

FULL ESTIMATED COST

INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, ANTE, AQUALINE, AQUASCI, BIOBUSINESS, BIOCOMMERCE, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, ...' ENTERED AT 18:36:38 ON 29 JUN 2005

74 FILES IN THE FILE LIST IN STNINDEX

Enter SET DETAIL ON to see search term postings or to view search error messages that display as 0* with SET DETAIL OFF.

=> s cytarabine or thioguanine or fludarabine or floxuridine or methotrexate or vincristine or vinblastine or paclitaxel or docetaxel or irinotecan
 22232 FILE ADISCTI

660	FILE ADISINSIGHT
3507	FILE ADISNEWS
518	FILE AGRICOLA
614	FILE ANABSTR
24	FILE ANTE
12	FILE AQUALINE
107	FILE AQUASCI
921	FILE BIOBUSINESS
173	FILE BIOCOMMERCE
745	FILE BIOENG
70329	FILE BIOSIS
1349	FILE BIOTECHABS
1349	FILE BIOTECHDS
20038	FILE BIOTECHNO
2132	FILE CABA
63374	FILE CANCERLIT
37382	FILE CAPLUS
160	FILE CEABA-VTB
69	FILE CEN
1230	FILE CIN
2355	FILE CONFSCI
23	FILE CROPB
40	FILE CROPU
21018	FILE DDFB
58423	FILE DDFU
26 FILES SEARCHED...	
10508	FILE DGENE
920	FILE DISSABS
21018	FILE DRUGB
1376	FILE DRUGMONOG2
60653	FILE DRUGU
604	FILE EMBAL
142055	FILE EMBASE
15970	FILE ESBIODBASE
1649	FILE FEDRIP
27	FILE FROSTI
19	FILE FSTA
1321	FILE GENBANK
148	FILE HEALSAFE
3830	FILE IFIPAT
1065	FILE IMSDRUGNEWS
983	FILE IMSPRODUCT
468	FILE IMSRESEARCH
5037	FILE JICST-EPLUS
25	FILE KOSMET
5610	FILE LIFESCI
80170	FILE MEDLINE
468	FILE NIOSHTIC
274	FILE NTIS
11	FILE NUTRACEUT
19	FILE OCEAN
30273	FILE PASCAL
1109	FILE PHAR
1231	FILE PHARMAML
57 FILES SEARCHED...	
19	FILE PHIC
2764	FILE PHIN
7067	FILE PROMT
1088	FILE PROUSDDR
10	FILE PS
5	FILE RDISCLOSURE
52266	FILE SCISEARCH
50	FILE SYNTHLINE
110850	FILE TOXCENTER
30192	FILE USPATFULL

2297 FILE USPAT2
 153 FILE VETB
 507 FILE VETU
 16 FILE WATER
 3822 FILE WPIDS
 30 FILE WPIFV
 3822 FILE WPINDEX

71 FILES HAVE ONE OR MORE ANSWERS, 74 FILES SEARCHED IN STNINDEX

L1 QUE CYTARABINE OR THIOGUANINE OR FLUDARABINE OR FLOXURIDINE OR METHOTREXATE OR VINCRISTINE OR VINBLASTINE OR PACLITAXEL OR DOCETAXEL OR IRINOTECAN

=> d rank

F1	142055	EMBASE
F2	110850	TOXCENTER
F3	80170	MEDLINE
F4	70329	BIOSIS
F5	63374	CANCERLIT
F6	60653	DRUGU
F7	58423	DDFU
F8	52266	SCISEARCH
F9	37382	CAPLUS
F10	30273	PASCAL
F11	30192	USPATFULL
F12	22232	ADISCTI
F13	21018	DDFB
F14	21018	DRUGB
F15	20038	BIOTECHNO
F16	15970	ESBIOBASE
F17	10508	DGENE
F18	7067	PROMT
F19	5610	LIFESCI
F20	5037	JICST-EPLUS
F21	3830	IFIPAT
F22	3822	WPIDS
F23	3822	WPINDEX
F24	3507	ADISNEWS
F25	2764	PHIN
F26	2355	CONFSCI
F27	2297	USPAT2
F28	2132	CABA
F29	1649	FEDRIP
F30	1376	DRUGMONOG2
F31	1349	BIOTECHABS
F32	1349	BIOTECHDS
F33	1321	GENBANK
F34	1231	PHARMAML
F35	1230	CIN
F36	1109	PHAR
F37	1088	PROUSDDR
F38	1065	IMSDRUGNEWS
F39	983	IMSPRODUCT
F40	921	BIOBUSINESS
F41	920	DISSABS
F42	745	BIOENG
F43	660	ADISINSIGHT
F44	614	ANABSTR
F45	604	EMBAL
F46	518	AGRICOLA
F47	507	VETU
F48	468	IMSRESEARCH
F49	468	NIOSHTIC
F50	274	NTIS

F51	173	BIOCOMMERCE
F52	160	CEABA-VTB
F53	153	VETB
F54	148	HEALSAFE
F55	107	AQUASCI
F56	69	CEN
F57	50	SYNTHLINE
F58	40	CROPU
F59	30	WPIFV
F60	27	FROSTI
F61	25	KOSMET
F62	24	ANTE
F63	23	CROPB
F64	19	FSTA
F65	19	OCEAN
F66	19	PHIC
F67	16	WATER
F68	12	AQUALINE
F69	11	NUTRACEUT
F70	10	PS
F71	5	RDISCLOSURE

=> file toxcenter and medline
 'AND' IS NOT A VALID FILE NAME
 Enter "HELP FILE NAMES" at an arrow prompt (=>) for a list of files that are available. If you have requested multiple files, you can specify a corrected file name or you can enter "IGNORE" to continue accessing the remaining file names entered.
 ENTER A FILE NAME OR (IGNORE):
 ENTER A FILE NAME OR (IGNORE):end

=> file medline		
COST IN U.S. DOLLARS	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	2.36	2.57

FILE 'MEDLINE' ENTERED AT 18:39:14 ON 29 JUN 2005

FILE LAST UPDATED: 29 JUN 2005 (20050629/UP). FILE COVERS 1950 TO DATE.

On December 19, 2004, the 2005 MeSH terms were loaded.

The MEDLINE reload for 2005 is now available. For details enter HELP RLOAD at an arrow prompt (=>). See also:

<http://www.nlm.nih.gov/mesh/>
http://www.nlm.nih.gov/pubs/techbull/nd04/nd04_mesh.html

OLDMEDLINE now back to 1950.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2005 vocabulary.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s cytarabine or thioguanine or fludarabine or floxuridine or methotrexate or vincristine or vinblastine or paclitaxel or docetaxel or irinotecan
 11453 CYTARABINE
 3239 THIOGUANINE
 1969 FLUDARABINE
 2853 FLOXURIDINE
 29781 METHOTREXATE
 19432 VINCRISTINE

```

11378 VINBLASTINE
10847 PACLITAXEL
2979 DOCETAXEL
2544 IRINOTECAN
L2 80170 CYTARABINE OR THIOGUANINE OR FLUDARABINE OR FLOXURIDINE OR METHO
TREXATE OR VINCRISTINE OR VINBLASTINE OR PACLITAXEL OR DOCETAXEL
OR IRINOTECAN

```

=> s l2 and (cytochalasin or dihyrocytochalasin or jasplakinolide or chondramide or latrunculin)

```

9843 CYTOCHALASIN
0 DIHYROCYTOCHALASIN
240 JASPLAKINOLIDE
6 CHONDRAMIDE
602 LATRUNCULIN
L3 669 L2 AND (CYTOCHALASIN OR DIHYROCYTOCHALASIN OR JASPLAKINOLIDE OR
CHONDRAMIDE OR LATRUNCULIN)

```

=> s l3 and cancer and (interphase or cytodieresis or cytokineses)

```

500869 CANCER
12639 INTERPHASE
10 CYTODIERESIS
14 CYTOKINESES
L4 0 L3 AND CANCER AND (INTERPHASE OR CYTODIERESIS OR CYTOKINESES)

```

=> s l3 and cancer and (interphase or cytodieresis or cytokinesis)

```

500869 CANCER
12639 INTERPHASE
10 CYTODIERESIS
3491 CYTOKINESIS
L5 2 L3 AND CANCER AND (INTERPHASE OR CYTODIERESIS OR CYTOKINESIS)

```

=> d 1-2 bib asb

'ASB' IS NOT A VALID FORMAT FOR FILE 'MEDLINE'

The following are valid formats:

The default display format is BIB.

```

ABS ---- AB
ALL ---- AN, DN, TI, CM, AU, CS, NC, SO, CY, DT, LA, FS, OS, EM,
ED, AB, ST, CT, NA, RN, CN, GEN
BIB ---- AN, DN, TI, CM, AU, CS, NC, SO, CY, DT, LA, FS, OS, EM, ED
CBIB --- AN, DN, TI, CM, AU, CS, NC, SO, CY, DT, LA, FS, OS, EM, ED
DALL --- ALL, delimited for post processing
IABS --- ABS, with a text label
IALL --- ALL, indented with text labels
IBIB --- BIB, indented with text labels
IND ---- ST, CT, NA, RN, CN, GEN
SAM ---- TI, CM, ST, CT, NA, RN, CN, GEN
TRI ---- TI, CM, ST, CT, NA, RN, CN, GEN
TRIAL -- TI, CM, ST, CT, NA, RN, CN, GEN
HIT ---- All fields containing hit terms
HITIND - IND
KWIC --- All hit terms plus 20 words on either side
OCC ---- List of display fields containing hit terms

```

Hit terms will be highlighted in all available fields except CM and PY.

To display a particular field or fields, enter the display field codes. For a list of display field codes, enter 'HELP DFIELDS' at an arrow prompt (=>). Examples of formats include: 'BIB'; 'AB'; 'SO,ST'. You may specify the format fields in any order, and the information will be displayed in the same order as the

format specification.

The same formats (except for HIT, HITIND, KWIC, and OCC) may be used with the DISPLAY ACC command to display the record for a specified Accession Number.

ENTER DISPLAY FORMAT (BIB):d 1-2 bib abs

'D' IS NOT A VALID FORMAT FOR FILE 'MEDLINE'

'1-2' IS NOT A VALID FORMAT FOR FILE 'MEDLINE'

The following are valid formats:

The default display format is BIB.

ABS ---- AB
ALL ---- AN, DN, TI, CM, AU, CS, NC, SO, CY, DT, LA, FS, OS, EM,
 ED, AB, ST, CT, NA, RN, CN, GEN
BIB ---- AN, DN, TI, CM, AU, CS, NC, SO, CY, DT, LA, FS, OS, EM, ED
CBIB --- AN, DN, TI, CM, AU, CS, NC, SO, CY, DT, LA, FS, OS, EM, ED
DALL --- ALL, delimited for post processing
IABS --- ABS, with a text label
IALL --- ALL, indented with text labels
IBIB --- BIB, indented with text labels
IND ---- ST, CT, NA, RN, CN, GEN
SAM ---- TI, CM, ST, CT, NA, RN, CN, GEN
TRI ---- TI, CM, ST, CT, NA, RN, CN, GEN
TRIAL -- TI, CM, ST, CT, NA, RN, CN, GEN
HIT ---- All fields containing hit terms
HITIND - IND
KWIC --- All hit terms plus 20 words on either side
OCC ---- List of display fields containing hit terms

Hit terms will be highlighted in all available fields except CM and PY.

To display a particular field or fields, enter the display field codes. For a list of display field codes, enter 'HELP DFIELDS' at an arrow prompt (=>). Examples of formats include: 'BIB'; 'AB'; 'SO,ST'. You may specify the format fields in any order, and the information will be displayed in the same order as the format specification.

The same formats (except for HIT, HITIND, KWIC, and OCC) may be used with the DISPLAY ACC command to display the record for a specified Accession Number.

ENTER DISPLAY FORMAT (BIB):end

=> s 15

500869 CANCER

12639 INTERPHASE

10 CYTODIERESIS

3491 CYTOKINESIS

L6 2 L3 AND CANCER AND (INTERPHASE OR CYTODIERESIS OR CYTOKINESIS)

=> d 1-2 bib abs

L6 ANSWER 1 OF 2 MEDLINE on STN

AN 2003189192 MEDLINE

DN PubMed ID: 12708486

TI Usefulness of combined treatment with mild temperature hyperthermia and/or tirapazamine in the treatment of solid tumors: its independence of p53 status.

AU Masunaga Shin-ichiro; Ono Koji; Takahashi Akihisa; Ohnishi Ken; Ohnishi Takeo; Suzuki Minoru; Nagata Kenji; Kinashi Yuko; Nagasawa Hideko; Uto Yoshihiro; Hori Hitoshi

CS Radiation Oncology Research Laboratory, Research Reactor Institute, Kyoto

University, Noda, Kumatori-cho, Sennan-gun, Osaka 590-0494..
smasuna@rri.kyoto-u.ac.jp

SO Cancer science, (2003 Jan) 94 (1) 125-33.
Journal code: 101168776. ISSN: 1347-9032.

CY Japan

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 200305

ED Entered STN: 20030424

Last Updated on STN: 20030514

Entered Medline: 20030513

AB Human head and neck squamous cell carcinoma cells transfected with mutant TP53 (SAS/mp53) or with neo vector as a control (SAS/neo) were inoculated subcutaneously into both hind legs of Balb/cA nude mice. Mice bearing the tumors received 5-bromo-2'-deoxyuridine (BrdU) continuously to label all proliferating (P) cells in the tumors. The mice then received tirapazamine (TPZ) with or without mild temperature hyperthermia (40 degrees C, 60 min) (MTH), gamma-ray irradiation with or without MTH and/or TPZ, cisplatin (CDDP) with or without MTH and/or TPZ, or **paclitaxel** (TXL) with or without MTH and/or TPZ. After each treatment, the tumors were excised, minced and trypsinized. The tumor cell suspensions thus obtained were incubated with a **cytokinesis** blocker (**cytochalasin-B**), and the micronucleus (MN) frequency in cells without BrdU labeling (i.e., quiescent (Q) cells) was determined by using immunofluorescence staining for BrdU. Meanwhile, 6 h after gamma-ray irradiation or 24 h after other cytotoxic treatments, tumor cell suspensions obtained in the same manner were used for determining the frequency of apoptosis in Q cells. The MN frequency and apoptosis frequency in total (P+Q) tumor cells were determined from the tumors that were not pretreated with BrdU. On the whole, gamma-ray irradiation and CDDP injection induced a higher frequency of apoptosis and lower frequency of MN in SAS/neo cells than SAS/mp53 cells. There were no apparent differences in the induced frequency of apoptosis and MN between SAS/neo and SAS/mp53 cells after TPZ or TXL treatment. MTH sensitized cells to TPZ-inducing cytotoxicity more markedly in SAS/mp53 and Q cells than in SAS/neo cells and total cells, respectively. In gamma-ray irradiation and CDDP treatment, the enhancement in combination with MTH and/or TPZ was more remarkable in SAS/mp53 cells and Q cells than in SAS/neo and total tumor cells, respectively. Also in the case of TXL treatment, the combination with MTH and/or TPZ induced a slightly greater enhancement effect in SAS/mp53 cells and Q cells. In view of the difficulty in controlling mutated p53 status tumors and intratumor Q cells, combination treatment with MTH and/or TPZ as a cooperative modality in **cancer** therapy is considered to have potential for controlling solid tumors as a whole.

L6 ANSWER 2 OF 2 MEDLINE on STN

AN 97206774 MEDLINE

DN PubMed ID: 9102841

TI [Significance of formation of micronuclei in SCC VII murine cells treated with various chemotherapeutic agents].
Znacaj formiranja mikronukleusa u murinim celijama SCC VII tretiranih razlicitim hemioterapijskim agensima.

AU Jeremic B; Sibamoto J; Abe M

CS Department of Oncology, Clinical-Hospital Centre, Kragujevac, Yugoslavia.

SO Srpski arhiv za celokupno lekarstvo, (1996 Jul-Aug) 124 (7-8) 169-74.

Journal code: 0027440. ISSN: 0370-8179.

CY Yugoslavia

DT Journal; Article; (JOURNAL ARTICLE)

LA Serbian

FS Priority Journals

EM 199704

ED Entered STN: 19970424

Last Updated on STN: 19970424

Entered Medline: 19970416

AB INTRODUCTION: The in vitro chemosensitivity testing aims at predicting the response of an individual tumour to chemotherapy choosing optimal agents for a particular patient. Among many chemosensitivity tests developed over the years [1-6], special emphasis was made on clonogenic assays that showed good use and correlation between laboratory and clinical data [7-9]. One of the assays used to predict the response to various anti-cancer modalities is the micronucleus assay using the cytokinesis-block [12-14]. This block is achieved by administration of **Cytochalasin-B** in order to prevent cytoplasmic, but not the nuclear, division. This leads to micronucleus formation which are counted in binuclear cells. Since there are only a few reports of the use of this assay in predicting chemosensitivity [13, 16], we explored the possibility of using this assay to predict chemosensitivity to various anti-cancer agents. MATERIAL AND METHODS: Exponentially growing SCC VII cells were treated with various concentrations of 11 anti-cancer agents: Mitomycin C, Doxorubicin (ADR), Epirubicin (EPI), Cisplatin, Carboplatin (CBDCA), Etoposide (VP-16), Vincristine, 5-fluorouracil, Methotrexate, Nimustine, and Dacarbazine for 1 hour. After that, **Cytochalasin-B** was added and dishes were incubated. After various time intervals, cells were fixed in situ and dried. Electron microscope was used to count the number of micronuclei (MN) in binucleate cells as well as multinucleate cells (MNC) in the total cell population. Cell survival was also evaluated by using the colony formation assay [18]. RESULTS: Maximal % of binucleate cells (BNC) was usually reached at 24-30 hours of culture, except for cells treated with ADR and EPI, in which it was reached at 30-72 hours (Figures 1 and 2). All drugs induced formation of micronuclei and dose-response curves for micronucleus frequency were obtained using the data at peak % BNC times. For all drugs, micronucleus frequency increased with concentration (Figure 3), but at the highest concentration used (considered to be overly toxic-Figure 4), the micronucleus frequency was rather lower. This decrease in micronucleus frequency was largely attributed to the decrease in % BNC. When the data at the highest concentrations of all drugs were excluded, a correlation was found between micronucleus frequency and surviving fraction ($r = 0.85$; $p < 0.001$) (Figure 5). DISCUSSION: Since micronucleus formation is a sign of chromosome damage that leads to cell death, we used this assay to evaluate chemosensitivity in 11 widely used anticancer agents. Although they can be classified according to mechanism of action as different class agents, they have in common the formation of micronuclei as a sign of cytotoxicity. Cell cycle arrest observed in some agents might be evaluated by assessing the delay in increase of BNC and MNC. The difference observed regarding cell cycle arrest suggested different mechanisms of its action. MN frequency was almost dose-dependent at lower concentrations, but at the highest concentrations, it obviously decreased, showing, therefore, some discrepancies with the data obtained when radiosensitivity was tested that way [14], probably due to extreme toxicity of agents. The optimal concentrations seem to be those providing a 20-80% surviving fraction. Another slight difference, when compared with similar radiosensitivity studies is a decrease with longer duration of culture observed in chemosensitivity testings. The reason for this difference is still unknown, but it emphasized the necessity for choosing the optimal duration of culture, probably that necessary for reaching maximal % BNC. This assay seems useful in predicting chemosensitivity of at least some tumour cells to various (appropriate) concentrations of various anti-cancer agents. However, new studies are warranted to further use of this assay, before testing it in clinical practice.

=> s 13 and "cell cycle"
1901827 "CELL"
195980 "CYCLE"
72783 "CELL CYCLE"
("CELL" (W) "CYCLE")

=> d 20-29 bib abs

L7 ANSWER 20 OF 29 MEDLINE on STN
 AN 91369816 MEDLINE
 DN PubMed ID: 1892755
 TI Resistance to the antimitotic drug estramustine is distinct from the multidrug resistant phenotype.
 AU Speicher L A; Sheridan V R; Godwin A K; Tew K D
 CS Department of Pharmacology, Fox Chase Cancer Center, Philadelphia, Pennsylvania 19111.
 SO British journal of cancer, (1991 Aug) 64 (2) 267-73.
 Journal code: 0370635. ISSN: 0007-0920.
 CY ENGLAND: United Kingdom
 DT Journal; Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals
 EM 199110
 ED Entered STN: 19911108
 Last Updated on STN: 19970203
 Entered Medline: 19911022

AB Following EMS mutagenesis, three estramustine (EM) resistant DU 145 human prostatic carcinoma cell lines were clonally selected by exposure to incrementally increasing concentrations of the drug. Although only low levels of resistance (approximately 3-fold) were attainable, this resistance was stable in the absence of continuous drug exposure. These EM-resistant clones (EMR 4,9,12) did not exhibit cross resistance to **vinblastine**, taxol, or adriamycin, and had collateral sensitivity to **cytochalasin B**. None of the lines had elevated expression of P-glycoprotein mRNA or glutathione S-transferase activity, suggesting a phenotype distinct from the classic multi-drug resistance phenotype. This conclusion was supported further by the observation that two MDR cell lines (FLC mouse erythroleukaemic and SKOV3 human ovarian carcinoma cells) showed sensitivity to EM. Fluorescent activated cell sorting analysis of the effects of EM on **cell cycle** traverse revealed that at EM concentrations up to 20 microM an increasing percentage of wild type cells were blocked in G2/M; no such effect occurred in EMR lines. Differential interference contrast microscopy was employed to study EM's effect on mitosis. EMR lines were able to form functional, albeit smaller, spindles at EM concentrations that resulted in chromosomal disorganisation and inhibition of mitotic progression in wild type cells. EMR lines were able to progress through mitosis and cytokinesis at the same rate as untreated cells. Tritiated EM was used to evaluate potential drug uptake/efflux mutations in EMR clones. EMR 4 and 9 incorporate less EM than wild type cells; however, they have significantly decreased cellular volumes. The initial efflux rate constants for EMR clones were greater than for wild type cells. Within 5 min greater than 70% of the drug was lost from resistant cells compared to a 50% loss by the wild type. Although the specific mechanisms of resistance have yet to be defined, the lack of collateral resistance to other MDR/anti-microtubule agents could serve as the basis for the clinical use of EM in combination chemotherapy.

L7 ANSWER 21 OF 29 MEDLINE on STN
 AN 91029202 MEDLINE
 DN PubMed ID: 2121336
 TI Cytotoxic effects of **cell cycle** phase specific agents: result of **cell cycle** perturbation.
 AU Kung A L; Zetterberg A; Sherwood S W; Schimke R T
 CS Department of Biological Sciences, Stanford University, California 94305.
 NC GM-14931 (NIGMS)
 SO Cancer research, (1990 Nov 15) 50 (22) 7307-17.
 Journal code: 2984705R. ISSN: 0008-5472.
 CY United States

DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199012
ED Entered STN: 19910208

Last Updated on STN: 19910208

Entered Medline: 19901207

AB Although agents which act in a **cell cycle** phase specific manner are commonly used in the clinic and in basic research, it is as yet unclear why these agents are cytotoxic. In this paper, we examine the cellular events associated with the cytotoxicity of aphidicolin and **vincristine** in CHO strain AA8 cells. Cell killing resulting from aphidicolin treatment was found to require a period of inhibition-free growth following removal of the drug and was associated with characteristic aberrant mitotic processes. The cytotoxic effects of aphidicolin could be antagonized by the concomitant inhibition of protein synthesis with cycloheximide in the period of DNA synthesis inhibition. Cell killing resulting from treatment with **vincristine** was associated with the aberrant segregation of nuclear material and the formation of multiple partial nuclei. **Vincristine** cytotoxicity was found to be antagonized by concomitant administration of cycloheximide or **cytochalasin D**. These data support a hypothesis that the cytotoxic effects of **cell cycle** phase specific agents do not derive directly from their biochemical actions per se. We propose that cell death results from processes that are evoked by dissociation of normally integrated **cell cycle** events, and that dissociation involves replicative/mitotic events in the case of aphidicolin and karyokinetic/nuclear reformation events in the case of **vincristine**.

L7 ANSWER 22 OF 29 MEDLINE on STN

AN 87301802 MEDLINE

DN PubMed ID: 2887300

TI Effects of cytoskeletal inhibitors on water proton relaxation time changes in unfertilized and fertilized sea urchin eggs.

AU Zimmerman S; Zimmerman A M; Cameron I L; Fullerton G D; Schatten H; Schatten G

NC HD-17087 (NICHD)

HD12913 (NICHD)

HD22902 (NICHD)

+

SO Cell biology international reports, (1987 Aug) 11 (8) 605-14.

Journal code: 7708050. ISSN: 0309-1651.

CY ENGLAND: United Kingdom

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals; Space Life Sciences

EM 198710

ED Entered STN: 19900305

Last Updated on STN: 19970203

Entered Medline: 19871020

AB Unfertilized and fertilized sea urchin eggs were used for pulsed proton NMR spin-lattice relaxation time (T1) measurements of cellular water. An 81% increase in T1 time at fertilization was largely explained by the accumulation of extracellular water in the perivitelline space. To assess the role of microtubule and actin filament assembly and disassembly, eggs were treated with drugs that are known to change these cytoskeletal elements (i.e., colchicine, taxol and **cytochalasin B**). Egg volume was also monitored in all studies to rule out the influence of water content changes on the observed T1 relaxation time changes. Neither assembly nor disassembly of microtubules changed the T1 relaxation time. The role of actin polymerization and depolymerization is discussed as a possible explanation for the observed **cell cycle** dependent water proton T1 relaxation time changes.

L7 ANSWER 23 OF 29 MEDLINE on STN
 AN 83004003 MEDLINE
 DN PubMed ID: 6126383
 TI The effects of cyclosporins on the cell cycle of
 t-lymphoid cell lines.
 AU Koponen M; Grieder A; Loor F
 SO Experimental cell research, (1982 Aug) 140 (2) 237-50.
 Journal code: 0373226. ISSN: 0014-4827.
 CY United States
 DT Journal; Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals
 EM 198212
 ED Entered STN: 19900317
 Last Updated on STN: 19950206
 Entered Medline: 19821203

L7 ANSWER 24 OF 29 MEDLINE on STN
 AN 81264473 MEDLINE
 DN PubMed ID: 7263771
 TI Effects of cytoskeletal disrupting agents on replication of bovine
 endothelium.
 AU Selden S C 3rd; Rabinovitch P S; Schwartz S M
 NC AG-01751 (NIA)
 HL-03174 (NHLBI)
 HL-18645 (NHLBI)
 SO Journal of cellular physiology, (1981 Aug) 108 (2) 195-211.
 Journal code: 0050222. ISSN: 0021-9541.
 CY United States
 DT Journal; Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals
 EM 198110
 ED Entered STN: 19900316
 Last Updated on STN: 19970203
 Entered Medline: 19811025

AB Colchicine and **vinblastine** inhibited endothelial cell migration
 but had no effect on the stimulation of replication seen at wound edges in
 cultures of endothelium at stationary density. This is in contrast to the
 effects of cytochalasins which inhibit both migration and replication at
 wound edges. Moreover, colchicine and **vinblastine** stimulated
 cell replication in the unwounded, confluent monolayer. This effect has
 kinetics similar to the stimulation of replication at a wound edge and is
 associated with an initial retraction of cell borders, leaving gaps
 between cells. **Cytochalasin** D inhibited the growth response to
 microtubule disrupting agents but did not prevent cell retraction.
 Stimulation of replication by microtubule disrupting agents was not
 dependent on serum but was synergistic with serum in cultures rinsed
 repeatedly with serum-free medium. The replication occurred prior to any
 cell loss. When, however, cells were allowed to complete mitosis, about
 one-half of the daughter cells detached from the monolayer so that there
 was no increase in cell density. We conclude that microtubule disrupting
 agents are the first agents found to be effective in stimulating growth of
 vascular endothelium at saturation density. These data further suggest
 that colchicine and **vinblastine** stimulate cell growth in a
 manner similar to wounding, where cell movement is a prerequisite to cell
 replication.

L7 ANSWER 25 OF 29 MEDLINE on STN
 AN 80067295 MEDLINE
 DN PubMed ID: 159787
 TI The cytoplasmic origin of variability in the timing of S phase in
 mammalian cells.
 AU Brooks R F
 SO Cell biology international reports, (1979 Dec) 3 (9) 707-16.

Journal code: 7708050. ISSN: 0309-1651.

CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198002
ED Entered STN: 19900315

Last Updated on STN: 19970203

Entered Medline: 19800226

AB The time at which S phase begins in mammalian cells is highly variable with respect to cell age. Evidence is presented that this variability does not arise because the initiation of DNA synthesis depends on the stochastic interaction of an initiator substance with a rare initiation site. Instead, the signal responsible for starting S phase must appear at random in the cytoplasm and may be transient.

L7 ANSWER 26 OF 29 MEDLINE on STN

AN 79180175 MEDLINE

DN PubMed ID: 286310

TI Production of a tissue-like structure by contraction of collagen lattices by human fibroblasts of different proliferative potential in vitro.

AU Bell E; Ivarsson B; Merrill C

SO Proceedings of the National Academy of Sciences of the United States of America, (1979 Mar) 76 (3) 1274-8.

Journal code: 7505876. ISSN: 0027-8424.

CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 197907
ED Entered STN: 19900315

Last Updated on STN: 19970203

Entered Medline: 19790725

AB Fibroblasts can condense a hydrated collagen lattice to a tissue-like structure 1/28th the area of the starting gel in 24 hr. The rate of the process can be regulated by varying the protein content of the lattice, the cell number, or the concentration of an inhibitor such as Colcemid. Fibroblasts of high population doubling level propagated in vitro, which have left the cell cycle, can carry out the contraction at least as efficiently as cycling cells. The potential uses of the system as an immunologically tolerated "tissue" for wound healing and as a model for studying fibroblast function are discussed.

L7 ANSWER 27 OF 29 MEDLINE on STN

AN 78082280 MEDLINE

DN PubMed ID: 620423

TI Inhibition of mycoplasma cell division by cytochalasin B.

AU Ghosh A; Maniloff J; Gerling D A

SO Cell, (1978 Jan) 13 (1) 57-64.

Journal code: 0413066. ISSN: 0092-9674.

CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 197803
ED Entered STN: 19900314

Last Updated on STN: 19900314

Entered Medline: 19780321

AB Mycoplasma gallisepticum has subcellular organelles which may function as a primitive "mitotic-like" apparatus. To investigate these further, we have studied the effects of cytochalasin B (CB) on M. gallisepticum. We found that CB inhibits cell division; this is the only procaryote thus far reported to be inhibited by CB. CB does not inhibit glucose or macromolecule precursor uptake. It stops cellular DNA synthesis, however, although RNA and protein synthesis continue (at a

reduced rate). CB removal results in a resumption of DNA synthesis, followed by cell division. There appears to be some degree of cell synchrony in this first division after CB removal. These results, together with morphological data, indicate that CB blocks at two points in the cell cycle: at the time "mitotic-like" structures are formed and at the time of cell division. It is suggested that the CB blocks may result from a disruption of actin-like protein structures required at these points in the cell cycle.

L7 ANSWER 28 OF 29 MEDLINE on STN

AN 77114120 MEDLINE

DN PubMed ID: 189934

TI Decreased adherence to the substrate in Rous sarcoma virus-transformed chicken embryo fibroblasts.

AU Weber M J; Hale A H; Losasso L

SO Cell, (1977 Jan) 10 (1) 45-51.

Journal code: 0413066. ISSN: 0092-8674.

CY ENGLAND: United Kingdom

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 197704

ED Entered STN: 19900313

Last Updated on STN: 19970203

Entered Medline: 19770425

AB Cell-substrate adherence in cultures of chicken embryo fibroblasts was examined by determining the number of cells which could be detached from the culture dish by a stream of medium. Transformed cells were significantly less adherent than their normal counterparts. In cultures infected with a mutant of Rous sarcoma virus which is temperature-conditional for transformation, adherence changed promptly following a temperature shift. This change did not require progression through the cell cycle. The transformation-specific decrease in adherence required new protein synthesis, but the restoration of adherence which occurred following a shift to the restrictive temperature could occur in the absence of new protein synthesis. Inhibitor experiments suggested the importance of microfilaments and perhaps microtubules in the changes in detachability. In addition, there was a positive correlation between levels of surface LETS protein and cell substrate adherence following a temperature shift, although it seems probable that the bulk of the surface LETS is neither necessary nor sufficient for maintenance of normal cell substrate adherence.

L7 ANSWER 29 OF 29 MEDLINE on STN

AN 77106339 MEDLINE

DN PubMed ID: 65050

TI Cell cycle-dependent inhibition of Kirsten Murine sarcoma-leukemia virus release by cytochalasin B.

AU Panem S

SO Virology, (1977 Jan) 76 (1) 146-51.

Journal code: 0110674. ISSN: 0042-6822.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 197703

ED Entered STN: 19900313

Last Updated on STN: 19970203

Entered Medline: 19770315

=>

---Logging off of STN---

=>

Executing the logoff script...

=> LOG Y

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

SESSION

FULL ESTIMATED COST

12.80

15.37

STN INTERNATIONAL LOGOFF AT 18:55:09 ON 29 JUN 2005